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# Respiratory Infection of Turkeys with *Listeria monocytogenes* Scott A

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SUMMARY. The pathogenesis of *L. monocytogenes* strain Scott A was studied by challenging day-old male turkey poults by air sac inoculation with tryptose phosphate broth containing $10^0$ cfu (control), $10^4$, $10^5$, and $10^6$ cfu (low challenge), or $10^7$ and $10^8$ cfu (high challenge) of the Scott A (serotype 4b) strain of *L. monocytogenes*. Mortality at 2 wk postinfection (PI) ranged from 25% for low challenge to 100% for high challenge ($P = 0.0001$). Gross and histopathological lesions were observed in heart, liver, spleen, lung, and bursa of Fabricius of mortalities at 4 days PI. *Listeria monocytogenes* challenge resulted in significantly decreased relative weight of the bursa of Fabricius and increased relative weight of the spleen, and *L. monocytogenes* was isolated by direct plating of liver, pericardium, brain, and both left and right stifle joint synovium (knee) cultures, as well as gall bladder, yolk sac, and cecal tonsil from transfer swabs onto *Listeria*-selective agar. Isolates were confirmed as positive using Gram stain, biochemical tests, and the Biolog system. High challenge resulted in confirmed *L. monocytogenes* isolation from 48% of left knee and 59% of right knee cultures. Low challenge resulted in isolation of *L. monocytogenes* from 11% of both left and right knee cultures. These results suggest that *L. monocytogenes* Scott A colonization of turkey knee synovial tissue can initiate in day-of-age poults and that *L. monocytogenes* Scott A can be invasive through air sac infection.

RESUMEN. Infección respiratoria con *Listeria monocytogenes* Scott A en pavos.

Se estudió la patogénesis de la cepa Scott A de *Listeria monocytogenes*, mediante el desafío por inoculación en el saco aéreo de pavipollos machos de un día de edad con desafíos bajos ($10^4$, $10^5$, y $10^6$ unidades formadoras de colonias -ufc-) o desafíos altos ($10^7$ y $10^8$ ufc) de la cepa de *L. monocytogenes* Scott A, serotipo 4b. La mortalidad dos semanas después del desafío varió entre el 25% para el desafío bajo y el 100% para el desafío alto ($P < 0.0001$). En las aves muertas 4 días post infección, se observaron lesiones macroscópicas e histopatológicas en corazón, hígado, bazo y bolsa de Fabricio. El desafío con *L. monocytogenes* resultó en una disminución significativa del peso relativo de la bolsa de Fabricio y en un aumento del peso relativo del bazo. Se aisló *L. monocytogenes* por siembra directa del hígado, pericardio, cerebro y del cultivo de liquido sinovial de ambas articulaciones de la rodilla, así como de hisopos de vesícula biliar, saco vitelino y tonsilas cecales transferidos directamente a agar selectivo para *Listeria*. Los aislamientos fueron confirmados como positivos utilizando tinción de Gram, pruebas bioquímicas y el sistema Biolog. El desafío alto resultó en el aislamiento de *L. monocytogenes* en el 48% de cultivos de la rodilla izquierda y en el 59% de los cultivos de la rodilla derecha. El desafío bajo resultó en el aislamiento de *L. monocytogenes* en el 11% de los cultivos de ambas rodillas. Estos resultados sugieren que la colonización por *L. monocytogenes* del tejido sinovial en pavos puede iniciarse en pavipollos de un día de edad y que la *L. monocytogenes* cepa Scott A puede ser invasiva a partir de la infección del saco aéreo.

Key words: *Listeria monocytogenes*, turkeys, respiratory infection, turkey osteomyelitis complex, food safety

Abbreviations: FAC = ferric ammonium citrate; FSIS = U.S. Department of Agriculture's Food Safety and Inspection Service; H&E = hematoxylin and eosin; PI = postinfection; TOC = turkey osteomyelitis complex; TPB = tryptose phosphate broth

*Listeria monocytogenes* is a ubiquitous bacterial pathogen that has had a long history as a cause of disease in animals, including poultry (42,61,89). While early food-borne epidemics resulting from infected poultry were reported as far back as 1961 (84), listeriosis in humans has only been recognized as an important food-borne disease since the 1980s, when a number of outbreaks were attributed to the contamination of poultry, particularly processed, ready-to-eat poultry products (81,82). Within recent years, dozens of recalls due to *Listeria* in poultry products have made the news, some having wide-ranging multistate and international involvement. In 1998, *L. monocytogenes* in turkey hot dogs and packaged meats was blamed for 21 deaths and more than 100 illnesses in 22 states (12) and resulted in the recall of 35 million pounds of product. In December 2000, the U.S. Department of Agriculture's Food Safety and Inspection Service (FSIS) investigators implicated turkey and chicken deli meats with a *L. monocytogenes* outbreak that covered 10 states and resulted in 29 illnesses, 4 deaths, and 3 miscarriages (69) and led to the recall of approximately 16.7 million pounds of ready-to-eat turkey and chicken (33). A voluntary recall of 14.5 million pounds of ready-to-eat meat and poultry products from a plant in Oklahoma affected delis and institutions nationwide as well as product shipped to Japan, Korea, Mexico, Puerto Rico, and the South Pacific in 2001 (34). An outbreak of listeriosis on the East Coast in 2002, during which 53 people became ill and at least 7 people died, led to a recall of nearly 30 million pounds of turkey and chicken products from two companies (35,36). In 2005, 39,000 pounds of smoked-turkey products produced in New York were recalled from distribution centers in Delaware, New Jersey, New York, and Florida (37). The number and magnitude of these recalls make it imperative to find the sources of *L. monocytogenes* contamination of ready-to-eat meat products.

A prevailing belief is that *L. monocytogenes* becomes a contaminant of the processing plant and of poultry products due to its widespread presence in the environment and the inability to properly sanitize processing equipment and workers' hands and gloves, rather than any intrinsic contamination of poultry (21,22,51,72,73,79) This hypothesis is supported by studies that have not detected *L. monocytogenes* on feathers or skin, in ceca or feces, or in the farm

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environment of flocks that were found to have contaminated product in the processing plant (15,21,52,72,73,79). A number of studies have reported that the incidence of *L. monocytogenes* contamination within poultry plants increases as the birds pass through the plant, with the greatest contamination occurring in the later stages of processing (21,38,39,72,73,86), which has led to the belief that environmental contamination of the processing facility is of more importance than contamination due to incoming birds.

However, it is also possible that *L. monocytogenes* contamination of turkey products and processing plants may be due to endogenous contamination of chronically infected birds that harbor bacteria within the joints and bone, where it can be protected from the host immune response within macrophages. Besides being an environmental contaminant, *L. monocytogenes* is also a facultative intracellular pathogen that can persist and multiply in cells of the monocyte–macrophage system (40,56) as well as in enterocytes, hepatocytes, and various protozoa (20,63). *Listeria monocytogenes* is known to infect joints, tendons, and bone (13,29,44,53,65) and is often associated with abscesses in animal infections (42). In one study, contamination on the hands and gloves of poultry handlers increased from 20% after chilling to 45.5% after cutting carcasses and to 59% when the parts were packaged (38). This observation may not only indicate that the processing plant is the source of contamination of poultry meat as was suggested but can also support the possibility that the synovial tissue can be a source of endogenous contamination during processing.

Turkey osteomyelitis complex (TOC) is the name given to a condition of healthy-appearing processed turkeys that have chronic, unapparent, soft-tissue and bone lesions that are only detected when carcasses with green discolored livers are cut open during mandatory inspection by the FSIS (1,7,14,17,28). In one study, green livers were found to occur in 0.13% of turkey hens and 0.65% of toms processed in the southeastern United States (5). A second study of 42,010 inspected turkeys from seven commercial farms in the same area found the average green-liver incidence to be 0.69%, (hens 0.14% and toms 1.04%) (70). The current inspection system results in nearly all of these turkeys being cut and downgraded; however, only about half of the turkeys with green livers actually have TOC lesions (4,7,14,28,66,70,87). More importantly, from a food-safety perspective, is the fact that TOC lesions are also present in some carcasses that do not have green livers (7,9,14) . At least 14 different species of bacteria, including pathogens such as *Salmonella* and *Yersinia* species, have been isolated from TOC lesions (7,14). It is likely that such unapparent contamination of bone and soft tissues of healthy-appearing turkeys may be an overlooked source of contamination of both poultry products and processing surfaces with pathogens of food-safety importance. Turkey osteomyelitis complex can be experimentally reproduced by injecting male turkeys with dexamethasone, a synthetic glucocorticoid, suggesting that the stressors of turkey production and selection for increased growth rate are affecting resistance to opportunistic bacterial infection in commercial turkey production (45,46,47,48,50).

For unknown reasons, *L. monocytogenes* serotype 4b has been the major serotype associated with human epidemics in both the United States and Europe (24,90). The serotype 4b strain used in this study, designated Scott A, is a human-epidemic isolate that was previously used to orally challenge 2-day-old chickens, resulting in 18% mortality. Most of the chickens eliminated the organism within 9 days, while long-term (28 days) infection was maintained in 1/10 challenged chicks (51). In both field infections (42) and experimental challenges (3,6,11,64), young poultry have been shown to be far more susceptible to listeriosis than are older birds. The present study is part of a larger effort to determine the sources of *L. monocytogenes* contamination of turkey products. The specific objectives of this study are to determine if a respiratory system challenge by air sac injection with *L. monocytogenes* Scott A is pathogenic in day-old poults and whether, under these experimental conditions, *L. monocytogenes* Scott A will colonize turkey knee synovial tissues.

## MATERIALS AND METHODS

**Challenge strain of *L. monocytogenes*.** The serotype 4b strain used in this study, designated Scott A, is a human-epidemic isolate that was obtained from the U.S. Food and Drug Administration Bacterial Physiology Branch (Cincinnati, OH). The inoculum was prepared by adding two loopsful of an overnight culture on blood agar to 100 ml of tryptose phosphate broth (TPB) and incubating for 2.5 hr in a 37 C shaking water bath. The culture was held overnight at 4 C while a standard plate count was made. Tenfold dilutions were then made in TPB based on the standard plate count.

**Experimental design.** Ninety male day-of-age commercial Hybrid turkey poults were placed in floor pens on wood shavings in two separate biosecure buildings, with nonchallenged controls housed separately and maintained using strict biosecurity. Birds were divided into six treatment groups, unchallenged controls, low challenge ($10^4$, $10^5$, and $10^6$ cfu), or high challenge ($10^7$ and $10^8$ cfu). The control, low-challenge, and high-challenge groups represented 24, 29, and 34 birds, respectively. A tuberculin syringe was used to inoculate the left cranial thoracic air sac at day of age with 100 μl TPB containing $10^4$, $10^5$, $10^6$, $10^7$, or $10^8$ cfu of *L. monocytogenes* Scott A . Unchallenged controls were inoculated with 100 μl TPB only.

Birds were allowed *ad libitum* access to water and a standard turkey starter diet that met or exceeded the recommendations of the National Research Council (71). Morbidity and mortality were monitored for 2 wk postchallenge. All dead birds, which were collected twice daily, and all necropsied birds were examined for myocardial lesions indicative of listeriosis and for airsacculitis/pericarditis using the following key, modified from that described by Piercy and West (76): 0, no inflammation; 1, opacity and thickening of the inoculated airsac; 2, mild airsacculitis and mild pericarditis; 3, moderate airsacculitis/pericarditis with spread to liver and/or abdominal cavity (perihepatitis/peritonitis); 4, severe fibrinous airsacculitis and severe pericarditis; 5, severe airsacculitis/pericarditis with spread to liver and/or abdominal cavity. Liver, heart, spleen, and bursa of Fabricius were excised and weighed.

On day 4 postchallenge, six unchallenged control birds were sacrificed and necropsied along with all poults that died that day. Birds and organs were weighed and tissues were taken for histopathology. The liver, pericardium, brain, and knee synovial tissues of every bird and the yolk sac, gall bladder, and cecal tonsils of selected birds were cultured with sterile transport swabs and directly plated on UVM modified *Listeria* enrichment media (Difco Laboratories, Detroit, MI) containing *Listeria* selective supplement (SR140E, Oxoid Limited, Ogdensburg, NY), moxalactam antimicrobial supplement (Becton Dickinson, Cockeyville, MD), and ferric ammonium citrate (FAC; Sigma-Aldrich Corp., St. Louis, MO). Synovial tissue isolates that were negative on direct plating were placed into Fraser broth tubes (Bio-Mérieux Vitek Inc., Lombard, IL) containing FAC for pre-enrichment and were replated on UVM plates. Isolated colonies were identified using Gram staining, hemolysis on Columbia blood agar (Remel, Microbiology Products, Lenexa, KS), biochemical tests (API Listeria Kit, Bio-Mérieux Vitek Inc, Hazelwood, MO) ,and the BioLog Microbial ID system (Biolog, Inc., Hayward, CA). Isolates were identified to the species level by the API *Listeria* kit according to the manufacturer's instructions. Isolates to be identified with the BioLog system were grown and processed as described in the system manual, inoculated into GP MicroPlates, and analyzed with the version DE, release 3.5, MicroLog database.

For histopathology, sections of liver, heart, spleen, bursa, lung, and brain were fixed in 10% neutral buffered formalin. Paraffin-embedded

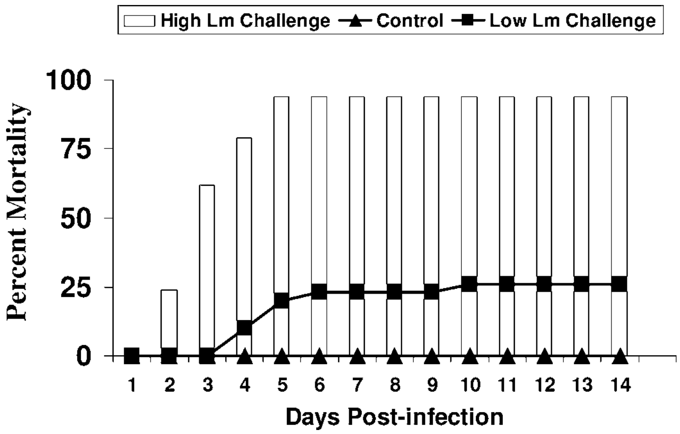


Fig. 1. Cumulative percent mortality of turkey poults 2 wk postinfection with respiratory challenge of *Listeria monocytogenes* (Lm) Scott A at day of age. The control, low Lm, and high Lm groups represented 24, 29, and 34 birds, respectively.

sections were cut at 5 μm, stained with hematoxylin and eosin (H&E) as well as Gram stain, and examined for histological lesions by a veterinary pathologist.

All research involving animals was evaluated and approved by the Institutional Animal Care and Use Committee of the University of Arkansas.

**Statistics.** All percentage data were subjected to arcsine transformation. Individual birds were used as the experimental unit and means were analyzed using the general linear models procedure of SAS software (80). Significant differences between treatments were separated using Duncan's multiple range test and, unless otherwise stated, a $P$ value of $\leq 0.05$ was considered significant.

## RESULTS

Respiratory challenge of day-old poults with *L. monocytogenes* Scott A resulted in mortality rates of 25% for low challenge and 100% for high challenge at 2 wk postchallenge ($P = 0.0001$). There was no mortality or pathology in unchallenged turkeys. Most of the mortality occurred between days 2 and 5 postchallenge (Fig. 1), with birds dying suddenly, without clinical signs. Some birds that died later in the experiment appeared drowsy and depressed and became dehydrated; however, most of the inoculated birds and all of the controls appeared healthy. Gross pathology included enlarged gall bladders and pale livers, some of which were also yellowish, mottled, or cooked in appearance. Occasionally, the gall bladder could be seen externally, pressing against the abdominal skin. Ruptured yolk sacs were common. Lungs appeared reddish-black and consolidated and hearts were swollen and surrounded by fluid. There was significantly more ($P = 0.05$) yolk sac infection in challenged birds when scored either on day 4 or at the end of the study. Mean airsacculitis scores were numerically but not significantly higher in challenged birds.

Histopathological lesions were observed in heart, liver, spleen, lung, and bursa of Fabricius of challenged turkeys. The myocardial lesions consisted of large infiltrations of mononuclear cells deep in the myocardium and were associated with gram-positive rods (Fig. 2). In the liver, focal infiltrations of mononuclear cells were small and scattered and were also associated with gram-positive rods (Fig. 3). Congestion and reticuloendothelial hyperplasia were prominent in the spleen and there was necrosis of scattered cells. Lymphocytes and mononuclear cells infiltrated areas surrounding bronchi in the lung. There was depletion of lymphocytes in follicles of the bursa of Fabricius (Fig. 4).

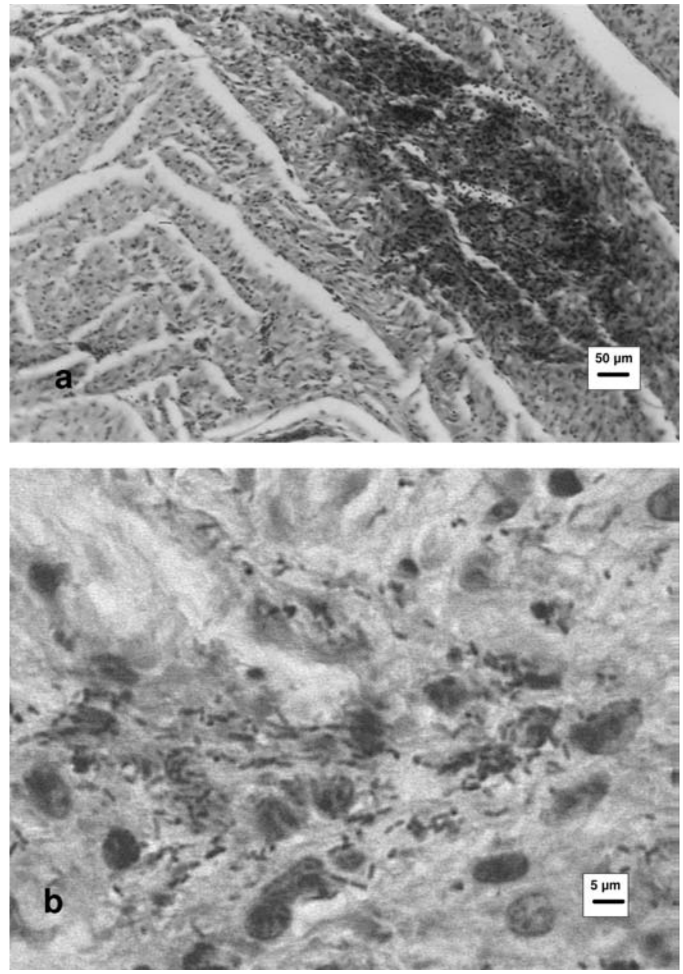


Fig. 2. Myocardial lesions of turkey poult 4 days after day-of-age airsac challenge with *Listeria monocytogenes*. (a) Necrotic foci deep in myocardium with infiltration of mononuclear cells (H&E). (b) Gram stain of myocardium showing association of mononuclear cells and gram-positive rods.

Body weight of *L. monocytogenes* challenged mortality at 4 days postinoculation (PI) was significantly decreased as compared with control birds ($P < 0.0001$; Table 1). The absolute heart weights were significantly lower in challenged birds, but the heart weight relative to body weight was not different. Both the absolute and the relative weights of the bursa of Fabricius were significantly decreased at 4 days PI with *L. monocytogenes* ($P \leq 0.0007$). The relative weights of the spleen were increased by *L. monocytogenes* challenge ($P \leq 0.05$), resulting in a bursa/spleen ratio that was lower in *L. monocytogenes* mortality as compared with nonchallenged controls ($P < 0.0001$; Table 1).

*Listeria monocytogenes* was isolated from liver, pericardium, brain, and knee synovial tissues that were directly plated on UVM *Listeria* agar but was not isolated from any control bird necropsied at 4 days PI (Table 2). *Listeria monocytogenes* was also isolated from the yolk sacs, gall bladders, and cecal tonsils of selected birds. High challenge resulted in confirmed *L. monocytogenes* isolation from 48% of left-knee and 59% of right-knee cultures. Low challenge resulted in isolation of *L. monocytogenes* from 11% of both left- and right-knee cultures (Table 2). Pre-enrichment in Fraser broth of both knee synovial tissue swabs that were negative on direct plating yielded an additional 16% confirmed *L. monocytogenes* isolation. High

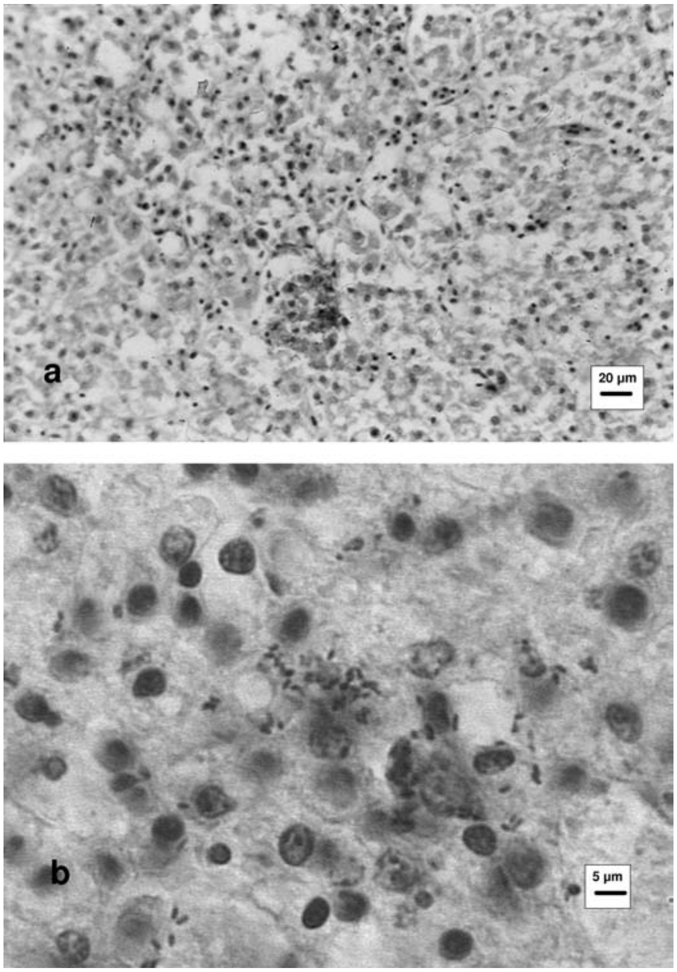


Fig. 3. Liver lesions of turkey poult 4 days after day-of-age airsac challenge with *Listeria monocytogenes*. (a) Small necrotic foci in liver with infiltration of mononuclear cells (H&E). (b) Gram stain of liver showing association of gram-positive rods and mononuclear cells.

challenge resulted in isolation of *L. monocytogenes* from 97% of the livers and pericardium and 88% of brains, while low challenge resulted in isolation rates of 78%, 67%, and 56% from liver, pericardium, and brain respectively (Table 2).

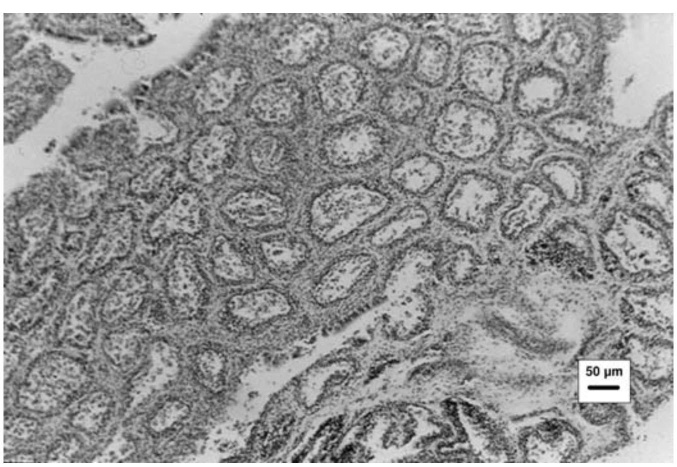


Fig. 4. Depletion of lymphocytes in follicle of the bursa of Fabricius of turkey poult 4 days after day-of-age airsac challenge with *Listeria monocytogenes* (H&E).

Table 1. Effect of low ($10^4$, $10^5$, and $10^6$ cfu) or high ($10^7$ and $10^8$ cfu) respiratory challenge with *Listeria monocytogenes* (Lm) strain Scott A on body weight and weights of heart, bursa, and spleen of turkey poults that died on day 4 postchallenge and six unchallenged control birds necropsied at 4 days postchallenge.$^A$

| | Unchallenged control ($n = 6$) | Low Lm challenge ($n = 2$) | High Lm challenge ($n = 5$) | $P$ value main effect mean |
|---|---|---|---|---|
| BWT (g) | $82.0 \pm 1.36^B$ | $54.50 \pm 3.10^C$ | $54.87 \pm 1.60^C$ | <0.0001 |
| Heart wt (g) | $0.54 \pm 0.03^B$ | $0.33 \pm 0.03^C$ | $0.43 \pm 0.0^C$ | 0.004 |
| Heart/BWT | $0.66 \pm 0.05$ | $0.64 \pm 0.14$ | $0.78 \pm 0.04$ | 0.19 |
| Bursa wt (g) | $0.15 \pm 0.01^B$ | $0.05 \pm 0.01^C$ | $0.05 \pm 0.01^C$ | <0.0001 |
| Bursa/BWT | $0.18 \pm 0.01^B$ | $0.11 \pm 0.01^C$ | $0.10 \pm 0.01^C$ | 0.0007 |
| Spleen wt (g) | $0.04 \pm 0.00^C$ | $0.07 \pm 0.01^{BC}$ | $0.09 \pm 0.02^B$ | 0.05 |
| Spleen/BWT | $0.05 \pm 0.00^C$ | $0.15 \pm 0.03^B$ | $0.16 \pm 0.03^B$ | 0.01 |
| Bursa/spleen | $3.90 \pm 0.46^B$ | $0.78 \pm 0.13^C$ | $0.81 \pm 0.22^C$ | <0.0001 |

$^A$Data represent the mean and standard error of the mean.
$^{BC}$Means within a row with no common superscript differ significantly as indicated.

## DISCUSSION

This study is the first report of experimental respiratory infection using an air sac challenge of turkeys with *L. monocytogenes*. Previous reports of oral and contact exposure of chickens (3,6) and intra-abdominal challenge of turkeys (64) indicate that young birds are far more susceptible to challenge than are older birds. This has also been true in our experience, as oral or respiratory challenge of 5-wk-old turkeys with *L. monocytogenes* Scott A results in minimal morbidity and mortality (49). The respiratory system is an important entry point for bacterial pathogens during confined poultry production on litter and has been shown to be important in studies of *L. monocytogenes* virulence (32,54,78). Respiratory challenge was shown to be the most consistent route in experimental infection of mice, hamsters, guinea pigs, rabbits, rhesus monkeys, and lambs with *L. monocytogenes* (54). These authors suggested that, in nature, *L. monocytogenes* infection most likely occurs through the respiratory tract and associated mucosal surfaces and conjunctiva. Respiratory challenge with *L. monocytogenes* has become a standard model for investigating pulmonary-host defense mechanisms and host–pathogen interactions in rats and mice (2,55,58,59,68).

Respiratory exposure to dust and pathogens in litter is a major cause of disease and condemnation in turkey production and the

Table 2. Effect of low ($10^4$, $10^5$, and $10^6$ cfu) or high ($10^7$ and $10^8$ cfu) respiratory challenge with *Listeria monocytogenes* (Lm) strain Scott A on isolation rate (percent) of Lm from liver, pericardium, brain, and left and right knee synovial tissues of mortalities and six nonchallenged control birds.$^A$

| | Unchallenged control ($n = 6$) % | Low Lm challenge ($n = 9$) % | High Lm challenge ($n = 29$) % | $P$ value main effect mean |
|---|---|---|---|---|
| Liver | $0 \pm 0^C$ | $78 \pm 15^B$ | $97 \pm 3^B$ | 0.0001 |
| Pericardium | $0 \pm 0^D$ | $67 \pm 17^C$ | $97 \pm 3^B$ | 0.0001 |
| Brain | $0 \pm 0^D$ | $56 \pm 18^C$ | $88 \pm 7^B$ | 0.0001 |
| Left knee | $0 \pm 0^C$ | $11 \pm 11^{BC}$ | $48 \pm 9^B$ | 0.01 |
| Right knee | $0 \pm 0^C$ | $11 \pm 11^C$ | $59 \pm 9^B$ | 0.001 |

$^A$Data represent the mean percent and standard error of the mean of Lm isolations from tissues of mortalities and from six nonchallenged controls.
$^{BCD}$Means within a row with no common superscript differ significantly as indicated.

respiratory route of infection is increasingly important as producers rely on built-up litter to defray the costs of both litter and litter disposal (8). While some surveys have failed to find *L. monocytogenes* in the litter of turkey houses (72) or broiler houses (62,73), litter contamination with *L. monocytogenes* has also been reported to be a persistent problem (25,26,27). Because *L. monocytogenes* is commonly found in fecal material, on plant materials, as well as in soil and water, and because it can survive for long periods of time under adverse conditions (31), it might be expected to have a sporadic presence in poultry litter and dust. Reports of *L. monocytogenes* outbreaks associated with wet litter conditions and environmental stress (19,57) suggest that, under the right circumstances, *L. monocytogenes* associated with litter can cause clinical disease in poultry.

While today listeriosis is no longer recognized as a disease problem in poultry production, historically it has been well documented as a sporadic disease of commercial poultry, including chickens, turkeys, and geese (16,23,42,60,74,75,83,84). More recently, reported outbreaks of listeriosis in chickens have involved high mortality and neurological signs (88) and encephalitis (18,19,57), and in two of these reports, excessively wet litter was associated with the outbreaks (19,57).

The pathology seen in the present study is consistent with earlier reports indicating that the lesions resembled those of an acute infection and the most conspicuous lesions are found in the heart, liver, spleen, and lungs (42,64,77,84). The observation that *L. monocytogenes* consistently decreases the weight of the bursa of Fabricius and causes lymphocytic depletion in bursal follicles has not been previously reported. Significantly decreased bursal/body weight ratios were also seen in challenge studies of 5-wk-old turkeys with *L. monocytogenes* Scott A (49). While immunity to *L. monocytogenes* has largely focused on T lymphocytes, Menon *et al.* (67) have demonstrated that *L. monocytogenes* can infect and kill B cells in a mouse model in order to initiate infection. As the bursa of Fabricius is the primary site of B-cell differentiation and maturation in avian species (85), these results suggest that *L. monocytogenes* can also invade and kill B cells in the turkey.

Gall bladder enlargement with concurrent isolation of *L. monocytogenes* was frequently seen in this experimental infection. In 1961, it was reported that inclusion of up to 40% bile did not inhibit *Listeria* multiplication (84). It was recently reported that *L. monocytogenes* can undergo extracellular replication to high numbers in the murine gall bladder in both diseased and asymptomatic individuals, suggesting that *L. monocytogenes* may be carried in the gall bladder in a manner similar to typhoid fever (43). Early literature concerning the epidemiology of listeriosis emphasized that "healthy carriers exist among human and animal populations and these appear to play a predominant role in transmission of the disease" (41,42,84). It has been hypothesized that active infection occurs when these healthy carriers are subjected to various forms of stress, and birds as well as other animals have been shown to be more susceptible to listeric infection when subjected to stress (10,41). Healthy carrier chickens were shown to be responsible for zoonotic transmission when *L. monocytogenes* conjunctivitis was acquired by poultry plant workers in 1944 (30).

The results of this study suggest that *L. monocytogenes* Scott A can be invasive through respiratory transmission and result in acute septicemia in turkeys, and that colonization of turkey synovial tissue can be initiated in day-old poults. Further study is needed to determine whether survivors of septicemic infections with *L. monocytogenes* and other food-borne pathogens can become chronic carriers with the potential of impacting the safety of turkey products.

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